Source: Finnish Medical Society Duodecim. Unstable angina pectoris. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2002 Apr 08.

Unstable angina pectoris

EBMG
08.04.2002

Contents

Objective

- To recognize angina pectoris that may be prodromal to acute infarction (acute coronary syndrome ACS) and to accompany the patient to a cardiac monitoring unit for active drug treatment or rapid revascularization (Level of Evidence=B; Evidence Summary available on the EBM Web site).

Definition

1. Recent (less than 1 - 2 months) angina pectoris
2. Accelerated angina pectoris
3. Angina pectoris at rest

Risk groups and clinical signs

- The presence of a marker (cardiac troponin T and I, CK-MBm) is the single most important predictor for future coronary events
    - Marker-positive patients are referred to angiography and revascularization
    - Marker-negatives are referred to exercise tolerance test.
- Unstable angina pectoris (UAP) is a heterogeneous group of diseases covering the range between stable AP and acute myocardial infarction (AMI).

- New (sudden) AP in a high-risk patient is always a serious condition.
- An aggravation in stable AP to unstable AP always necessitates a reassessment of risk and often a change in the line of treatment.
- There may not always be pain rather the main symptom is a decrease in exercise tolerance (sudden decrease in physical fitness) or acute left ventricle failure.
- In the ECG a ST segment depression precedes the pain. Symptomless (silent) ischaemia in a patient at risk is a significant finding. Ischaemia may not always be visible in ECG. An ECG registered while the patient has pain is invariably valuable.
- The border between UAP and T-wave infarction (non-Q infarction) is shifting. For example, very proximal occlusion in the left anterior descending artery (LAD) causes a symmetric T inversion in chest leads. Elevation of myocardial markers indicates that the patient has an infarction.

Treatment

- Treatment is normally carried out in a cardiac monitoring unit.
- Pharmacological treatment should be started in the first point of care. -The mildest form (recent angina) can be treated in a health care centre ward under careful monitoring. Remember the risk of MI. The risk diminishes with time as the angina stabilizes.

Anti-ischaemic and antithrombotic treatment

- ASA 250 mg (chewable) first. Thereafter 100 mg/day, unless there are contraindications.
- Oxygen
- Nitrate infusion (Level of Evidence=D; Evidence Summary available on the EBM Web site) for 24 - 36 hours (See related EBM Guideline: **Nitrate infusion in angina pectoris and myocardial infarction** available on the EBM Web site). Systolic blood pressure should be lowered by 10 - 15 mmHg and always to a level below 150 mmHg.
- Beta-blocker (metoprolol or atenolol). Heart rate should be 50 - 70 beats/min and systolic pressure below 150 mmHg.
- Low-molecular-weight (LMW) heparin (Level of Evidence=A; Evidence Summary available on the EBM Web site) (e.g. dalteparin 100 - 120 IU x 2 daily for one week) is given simultaneously with ASA. The treatment can be continued with half the dose for about 1 month. UAP patients with an elevated troponin T concentration derive the greatest benefit from the treatment (LMWH + ASA).
- Pharmacotherapy and invasive treatment do not exclude one another.

Continuing the treatment

- High-risk patients
- Unstable angina and ischaemia on ECG or elevated myocardial markers (Level of Evidence=A; Evidence Summary available on the EBM Web site) acute left ventricle failure (lung oedema, mitral regurgitation, hypotension)
    - immediate angiography and revascularization. While waiting for the procedure the thrombosis can be stabilized with an i.v. GPIIb/IIIa inhibitor in addition to ASA and LMW heparin. (Fibrinolytic treatment has not effect on a thrombocytic block.
- No symptoms or signs of ischaemia on ECG
    - symptom-limited exercise test performed within 2 - 4 days.

    - If the patient has symptoms or signs of ischaemia during the exercise test or signs in ECG at a low pulse-pressure product, refer immediately to angiography.
    - In case of no symptoms or signs of ischaemia during light exercise or no signs in ECG, or if they occur only with a high pulse-pressure product, begin conservative treatment and elimination of risk factors. Prophylaxis can be intensified by adding clopidogrel to aspirin.
- Thrombolytic therapy or immediate percutaneous transluminal angioplasty (PTA) (during which a stent can be inserted) is indicated if ECG reveals a transmural injury. See article on revascularization (See related EBM Guideline: **Angina pectoris and coronary artery disease (CAD)** available on the EBM Web site). After the insertion of a stent, clopidogrel is used in combination with ASA for one month.

Organizing treatment

- UAP is a serious but often curable syndrome. A well-organized care pathway ensures that the appropriate treatment can be given rapidly.

Related evidence

- Intravenous heparin combined with aspirin is probably effective compared to aspirin alone in reducing myocardial infarction or death, but the number of patients studied is too small for statistical significance (Level of Evidence=C; Evidence Summary available on the EBM Web site).

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Author(s): Editors
Article ID: P04054 (004.058)